What is claimed is:

1. A method for identifying a compound which modulates an interaction between a first and a second polypeptide comprising:

(a) contacting a cell having a first polypeptide comprising a binding portion of a KRC polypeptide and a second polypeptide comprising a binding portion of a polypeptide selected from the group consisting of: Jun, GATA3, SMAD, or Runx2 in the presence and the absence of a test compound; and

(b) determining the degree of interaction between the first and the second polypeptide in the presence and the absence of the test compound,

to thereby identify a compound which modulates an interaction between a first and a second polypeptide.

2. The method of claim 1, wherein the first polypeptide comprises at least one KRC zinc finger domain.

3. The method of claim 1, wherein the second polypeptide is a c-Jun polypeptide.

4. The method of claim 1, wherein the second polypeptide is a SMAD2 polypeptide.

5. The method of claim 1, wherein the second polypeptide is a SMAD3 polypeptide.

6 The method of claim 1, wherein the first polypeptide is derived from an exogenous source.

7. The method of claim 1, wherein the second polypeptide is derived from an exogenous source.

8. The method of claim 1, wherein the cell is a yeast cell.

9. The method of claim 8, wherein determining the ability of the test compound to modulate the interaction of the first polypeptide and the second polypeptide comprises determining the ability of the compound to modulate growth of the yeast cell on nutritionally selective media.

10 The method of claim 8, wherein determining the ability of the test compound to modulate the interaction of the first polypeptide and the second polypeptide comprises determining the ability of the compound to modulate expression of a reporter gene in the yeast cell.

11. The method of claim 1, wherein determining the ability of the test compound to modulate the interaction of the first polypeptide and the second polypeptide comprises determining the ability of the test compound to modulate the coimmunoprecipitation of the first polypeptide and the second polypeptide.

12. The method of claim 1, wherein determining the ability of the test compound to modulate the interaction of the first polypeptide and the second polypeptide comprises determining the ability of the test compound to modulate signaling via a signal transduction pathway involving KRC in the cell.

13 The method of claim 12, wherein at least one of TNFα production, IL-2 production, AP-1 activity, Ras and Rac activity, actin polymerization, ubiquitination of AP-1, ubiquitination of TRAF, ubiquitination of Runx2, degradation of c-Jun, degradation of c-Fos degradation of SMAD, degradation of Runx2, degradation of GATA3, GATA3 expression, Th2 cell differentiation, Th2 cytokine production, IgA production, GLα transcription (Igα chain germline transcription), and/or osteocalcin gene transcription is measured.

14. The method of claim 12, wherein ubiquitination or degradation of c-fos, c-Jun, SMAD3, GATA3 or Runx2 is measured.

15. The method of claim 12, wherein AP-1, TRAF2 or Runx2 ubiquitination is measured.

16. The method of claim 1, wherein the binding of first and second polypeptide is inhibited.

17. The method of claim 1, wherein the binding of first and second polypeptide is stimulated.

18. A method of identifying a compound that modulates a mammalian KRC biological activity comprising:

(a) contacting cells deficient in KRC or a molecule in a signaling pathway involving KRC with a test compound; and

(b) determining the effect of the test compound on the KRC biological activity, the test compound being identified as a modulator of the biological activity based on the ability of the test compound to modulate the biological activity in the cells deficient in KRC or a molecule in a signaling pathway involving KRC to thereby identify a compound that modulates a mammalian KRC biological activity.

19. The method of claim 18, wherein the biological activity of KRC is selected from the group consisting of modulation of: modulation of a TGFβ signaling pathway, modulation of ubiquitination of AP-1, modulation of ubiquitination of TRAF, modulation of ubiquitination of Runx2, modulation of the degradation of c-Jun, modulation of the degradation of c-Fos, modulation of degradation of SMAD, modulation of degradation of Runx, modulation of degradation of GATA3, modulation of GATA3 expression, modulation of Th2 cell differentiation, modulation of Th2 cytokine production, modulation of IgA production, modulation of GLα transcription, or modulation of osteocalcin gene transcription.

20. The method of claim 18, wherein the cells are in a non-human animal deficient in KRC or a molecule in a signal transduction pathway involving KRC and the cells are contacted with the test compound by administering the test compound to the animal.

21. A method of identifying compounds useful in modulating a biological activity of mammalian KRC comprising:

a) providing an indicator composition comprising mammalian KRC or a molecule in a signal transduction pathway involving KRC;

b) contacting the indicator composition with each member of a library of test compounds;

c) selecting from the library of test compounds a compound of interest that modulates a biological activity of KRC or the molecule in a signal transduction pathway involving KRC; to thereby identify a compound that modulates a biological activity of mammalian KRC, wherein the biological activity of KRC is selected from the group consisting of: modulation of a TGFβ signaling pathway, modulation of ubiquitination of AP-1, modulation of ubiquitination of TRAF, modulation of ubiquitination of Runx2, modulation of the degradation of c-Jun, modulation of the degradation of c-Fos, modulation of degradation of SMAD, modulation of degradation of Runx, modulation of degradation of GATA3, modulation of GATA3 expression, modulation of Th2 cell differentiation, modulation of Th2 cytokine production, modulation of IgA production, modulation of GLα transcription, and modulation of osteocalcin gene transcription.

22. The method of claim 21, wherein the indicator composition is a cell that expresses KRC, and at least one molecule selected from the group consisting of: c-Jun, c-Fos, AP-1, GATA3, SMAD, and Runx2 protein.

23. The method of claim 21, wherein the indicator composition is a cell free composition.

24. A method for modulating the expression and/or biological activity of a KRC polypeptide in a subject, comprising contacting an immune cell from the subject with a compound that modulates the expression and/or biological activity of a KRC polypeptide in the immune cell, such that the expression and/or biological activity of the KRC polypeptide in the subject is modulated, wherein the biological activity of KRC is selected from the group consisting of: modulation of a TGFβ signaling pathway, modulation of ubiquitination of AP-1, modulation of ubiquitination of TRAF, modulation of ubiquitination of Runx2, modulation of the degradation of c-Jun, modulation of the degradation of c-Fos, modulation of degradation of SMAD, modulation of degradation of Runx, modulation of degradation of GATA3, modulation of GATA3 expression, modulation of Th2 cell differentiation, modulation of Th2 cytokine production, modulation of IgA production, modulation of Glα transcription, and modulation of osteocalcin gene transcription.

25. The method of claim 24, wherein the step of contacting occurs *in vivo*.

26. The method of claim 24, wherein the step of contacting occurs *in vitro*.

27. The method of claim 24, wherein the cell is selected from the group consisting of: a T cell, a B cell, and a macrophage.

28. The method of claim 24, wherein KRC activity is enhanced.

29. The method of claim 24, wherein KRC activity is inhibited.

30. The method of claim 28, wherein the agent is selected from the group consisting of: a nucleic acid molecule encoding a polypeptide comprising a biologically active KRC domain, a polypeptide comprising a biologically active KRC domain, and a small molecule KRC agonist.

31. The method of claim 29, wherein the agent is selected from the group consisting of: an intracellular antibody, a nucleic acid molecule that is antisense to a nucleic acid

molecule encoding KRC, a KRC siRNA molecule, a dominant negative KRC molecule, and a small molecule KRC antagonist.

32. A method for modulating the interaction between a KRC molecule and a KRC-binding partner comprising contacting an immune cell with an agent that modulates the interaction between KRC and a KRC-binding partner in the immune cell such that the interaction between KRC and a KRC-binding partner is modulated, wherein the KRC-binding partner is selected from the group consisting of c-Jun, GATA3, SMAD, or Runx2.

33. The method of claim 32, wherein the step of contacting occurs *in vivo*.

34. The method of claim 32, wherein the step of contacting occurs *in vitro*.

35. The method of claim 32, wherein the interaction between a KRC molecule and a KRC-binding partner molecule is inhibited.

36. The method of claim 32 wherein the agent is selected from the group consisting of: an intracellular antibody, a nucleic acid molecule that is antisense to a TRAF molecule, a nucleic acid molecule that is antisense to a c-Jun molecule, a nucleic acid molecule that is antisense to a KRC molecule, a nucleic acid molecule that is antisense to a c-Jun molecule a nucleic acid molecule that is antisense to a GATA3 molecule, a nucleic acid molecule that is antisense to a SMAD molecule, a nucleic acid molecule that is antisense to a RUNX2 molecule, a dominant negative KRC molecule, a dominant negative c-Jun molecule, a dominant negative GATA3 molecule, a dominant negative SMAD molecule, and a dominant negative Runx2 molecule.

37. The method of claim 32, wherein the portion of KRC that interacts with c-Jun, GATA3, SMAD, or Runx2 comprises amino acid residues 204-1055 of KRC.

38. The method of claim 32, wherein the agent that modulates the interaction between a KRC molecule and a KRC- binding partner molecule is useful for the treatment of an autoimmune disease in a subject.

39. The method of claim 32, wherein the agent that modulates the interaction between a KRC molecule and a KRC- binding partner molecule is useful for the treatment of an malignancy in a subject.

40. The method of claim 32, wherein the agent that modulates the interaction between a KRC molecule and a KRC- binding partner molecule is useful for the treatment of a metabolic bone disease in a subject.

41. The method of claim 38, wherein the autoimmune disease is selected from the group consisting of: systemic lupus erythematosus; rheumatoid arthritis; goodpasture's syndrome; Grave's disease; Hashimoto's thyroiditis; pemphigus vulgaris; myasthenia gravis; scleroderma; autoimmune hemolytic anemia; autoimmune thrombocytopenic purpura; polymyositis and dermatomyositis; pernicious anemia; Sjögren's syndrome; ankylosing spondylitis; vasculitis, multiple sclerosis, inflammatory bowel disease, ulcerative colitis, Crohn's disease, and type I diabetes mellitus.

42. The method of claim 39, wherein the malignancy is selected from the group consisting of: acute lymphoblastic leukemia; acute myeloid leukemia; adrenocortical carcinoma; AIDS-related lymphoma; B cell chronic lymphocytic leukemia; cancer of the bile duct; bladder cancer; bone cancer, osteosarcomal malignant fibrous histiocytomal brain stem gliomal brain tumor; breast cancer; bronchial adenomas; carcinoid tumors; adrenocortical carcinoma; central nervous system lymphoma; cancer of the sinus, cancer of the gall bladder; gastric cancer; cancer of the salivary glands; cancer of the esophagus; neural cell cancer; intestinal cancer (*e.g.,* of the large or small intestine); cervical cancer; colon cancer; colorectal cancer; cutaneous T-cell lymphoma; B-cell lymphoma; T-cell lymphoma; endometrial cancer; epithelial cancer; endometrial cancer; intraocular melanoma; retinoblastoma; hairy cell leukemia; liver cancer; Hodgkin's disease; Kaposi's sarcoma; acute lymphoblastic leukemia; lung cancer; non-Hodgkin's lymphoma; melanoma; multiple myeloma; neuroblastoma; prostate cancer; retinoblastoma; Ewing's sarcoma; vaginal cancer; Waldenstrom's macroglobulinemia; adenocarcinomas; ovarian cancer, chronic lymphocytic leukemia, pancreatic cancer; and Wilm's tumor.

43. The method of claim 40, wherein the metabolic bone disease is selected from the group consisting of: osteoporosis, osteomalacia, skeletal changes of hyperparathyroidism and chronic renal failure (renal osteodystrophy) and osteitis deformans (Paget's disease of bone).

44. A method for inhibiting a neoplasia in a subject, comprising contacting a tumor cell from the subject with a compound that modulates the expression and/or biological activity of KRC in the tumor cell such that the neoplasia in the subject is inhibited.

45. The method of claim 44, wherein the neoplasia is a B cell chronic lymphocytic leukemia

46. A non-human animal, in which the gene encoding the KRC gene is misexpressed.

47. The animal of claim 46, wherein the animal is a transgenic animal.

48. The animal of claim 47, wherein the transgenic animal is a mouse.

49. The animal of claim 46, wherein the KRC gene is disrupted by removal of DNA encoding all or part of the KRC protein.

50. The animal of claim 49, wherein the animal is homozygous for the disrupted gene.

51. The animal of claim 49, wherein the animal is heterozygous for the disrupted gene.

52. The animal of claim 46, wherein the animal is a transgenic mouse with a transgenic disruption of the KRC gene.

53. The animal of claim 52, wherein the disruption is an insertion or deletion.

54. A transgenic mouse comprising in its genome an exogenous DNA molecule that functionally disrupts a KRC gene of said mouse, wherein the mouse exhibits a phenotype characterized by impaired Th2 cell development, decreased Th2 cytokine production, impaired TGFβR signaling in B cells, decreased IgA secretion and decreased transcription of the GLα gene, relative to a wildtype mouse.